converted into absolute value, as oppose to a gradual rise in the number of Mon2/ml, the number of mDC2/ml remain gradually decreased with the progress of plaque vulnerability. Plasma levels of Lp-PLA2, PTX3, FABP4 and myeloperoxidase, all of which reflected coronary plaque vulnerability, were positively correlated with the number of Mon2/ml, but negatively with the number of mDC2/ml.
Conclusions: Circulating subsets of mDC2 and Mon2 appear to be promosing markers of plaque stabilization and rupture.

GW25-e0795

Relationship between Stress Hyperglycemia and in hospital Mortality and Complications in Patients with Acute Myocardial Infarction

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Objectives: To investigate the effect of stress hyperglycemia on the mortality and cardiovascular events in patients with acute myocardial infarction (AMI).
Methods: The study covered 1226 patients with the diagnosis of acute myocardial Infarction admitted in CCU from January 2010 to December 2012 in the First Affiliate Hospital of Dalian University. Based on the level of fasting blood glucose (FBG), all patients were grouped into two groups: high blood glucose group (HBG group, ≥7mmol/L) and non-high blood glucose group (non-HBG group, <7mmol/L). Based on the history of diabetes were grouped into non-diabetes mellitus group (NDM group) and diabetes mellitus group (DM group). According to the level of FBG, patients within DM and NDM group were further divided into groupA (FBG<7.0mmol/L), groupB (7.0mmol/L≤FBG<9.0mmol/L), groupC (9.0mmol/L≤FBG<11.1mmol/L),groupD (FBG≥11.1mmol/L), respectively. Compare the differences of the mortality and the rate of acute heart failure, cardiogenic shock and main arrhythmias among these groups.
Results: The rate of high FBG is 34.1% in all AMI, among 37% patients without history of diabetes mellitus. Compare with the 808 non-HBG groups patients, the 418 HBG group patients had higher mortality (9.1% vs 2.1%) and higher rate of acute heart failure (37.8% vs 21.2%), cardiogenic shock (7.9% vs 0.7%) and main arrhythmias (25.6% vs 16.5%) (all P<0.01). The mortality (6.4% vs 3.7%) and the rate of acute heart failure (35.9% vs 23%) and cardiogenic shock (6.6% vs 1.7%) are greater in the 362 DM group patients than the 864 NDM group (P<0.05).Among the NDM group, the mortality and cardiovascular complications increased incrementally with the increasing of FBG. Compared with groupA, the mortality and the rate of acute heart failure, cardiogenic shock and main arrhythmias of groupB, groupC, groupD are significantly higher (all P<0.05). Compared with groupB, that mortality of groupD increase obviously (30% vs 2.0%, P<0.01). In the DM group, the mortality showed no significant differences with the increased FBG levels, but the rate of acute heart failure increased incrementally as FBG reached 9 mmol/L compared with group A (P<0.01). The mortality of impaired fasting glucose (IFG) patients is similar to the patients with normal FBG (2.4% vs 1.9%,P>0.05), and significantly lower than the patients with FBG≥7.0mmol/L (11.5% vs 2.4%,P<0.01).
Conclusions: The stress hyperglycemia could be used as a predictor of inhospital mortality and cardiovascular events for patients with AMI. The elevation of FBG increases the mortality and the incidence of acute heart failure, cardiogenic shock, and main arrhythmias of patients with AMI. But the effects were not consistent between DM and NDM patients. In NDM patients, as the FBG level increased, the mortality increased significantly, but this results was not obtained in DM group. In DM group, the incidence of acute heart failure was significantly increased as the FBG level increased.

GW25-e2360

The changes of PPARγ and EPCs in patients with ACS complicated with diabetes mellitus and the effect of Irbesartan on them

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Objectives: To observe the level of PPARγ and EPCs in patients suffered from ACS combined with Type-2 diabetes mellitus (T2DM) and ACS without T2DM, analysis the relationship between PPARγ, EPCs and ACS, T2DM, furthermore explore the influence of Irbesartan on them to study the mechanisms of endothelial protection beyond the antihypertensive effect of Irbesartan, hoping to find a new treatment method for ACS combined with T2DM.
Methods: 102 patients suffered from ACS was enrolled into our study, of which 52 of them combined with T2DM (ACSDM group) and the other 50 patients only suffered from ACS. Meanwhile, we selected 30 patients without coronary heat disease and T2DM as the control group. To all patients, blood was drew when they were enrolled to detect the level of PPARγ and $CD34^+/CD309^+$ EPCs. All basic clinic data, CAG and then Gensini score were compared among all groups. ACSDM group patients were divided into A, B two group randomly. Patients, who were in A group, were treated with Irbesartan 75mg po qd excepting conventional treatment of coronary heart and diabetes. Patients, who wre in B group, were treated without Irbesartan. Blood was drew again after 12 weeks. The level of PPARγ was detected by enzyme-linked immunosorbent method, meanwhile EPCs was detected by Flow cytometry instrument. We analyed datas of each group by applicating of SPSS17.0.
Results: (1) The basic clinic data among each group, such as age, sex, body mass index (BMI), smoking history, blood pressure, stain use, TG, HDL-CH, TC, LDL-CH, blood urea nitrogen, creatinine were no difference (*P* all>0.05). CHD history, STEMI/NSTEMI were no difference in ACS group and ACSDM group (*P* all>0.05). FPG, HbA1c in the ACSDM group were significantly much more than those in the ACS group and control group (*P* <0.05). (2) The levels of PPARγ of ACS group and ACSDM group, when compared with control group (295.56±25.06 ng/L), were decreased significantly (*P* all <0.05), and the ACSDM group was much lower than the ACS group (*P*<0.05). (3) EPCs levels in ACS and ACSDM group were also significantly lower, when compared with control group (0.0584±0.0142%, and the ACSDM group was much lower than the ACS group (*P*<0.05). 4 Gensini points in ACSDM group was much higher than that of the ACS group (42.24±25.46 VS. 30.26±18.35, *P*<0.01). 5 Levels of PPARγ and EPCs were significatly correlated positively (r=0.658, *P*<0.01). Gensini scores of patients with ACS was correlated negatively with both level of PPARγ (r=-0.484,*P*<0.05), and EPCs (r=-0.435,*P*<0.05). Levels of PPARγ and EPCs in combined group were significantly correlated positively (r=0.558, *P*<0.01). 6 After 12 weeks intervention of irbesartan, the level of PPARγ of A group increased significantly compared with pre-treatment levels [(226.17±25.95) ng/L VS. (234.55±26.20) ng/L, *P*<0.01], so did the level of EPCs [(0.0284±0.0111) % VS. (0.0311±0.0102) %, *P*<0.01]. There was a significant positive relationship between them (r=0.767, *P*<0.01). The level of PPARγ and EPCs of B group had on statistical difference after 12 weeks conventional treatment (*P* all>0.05).
Conclusions: The level of PPARγ, EPCs of patients with ACS are siganificantly lower than those of control group, being much lower in ACSDM group. PPAR γ and EPCs level both decrease with the increasing of the degree of coronary artery stenosi. Irbesartan, can improve the levels of PPARγ and EPCs in patients with ACS and T2DM, which probably become a new targets for therapeuty.

GW25-e3089

Bleeding outcomes in low-mediate risk acute coronary syndrome patients receiving stenting predicted by adenosine diphophate induced platelet aggregation after initiation of clopidogrel: 6 months follow-up

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Objectives: The correlation of enhanced platelet responder to clopidogrel with bleeding outcome and entry-site complication has rarely been characterized in China population undergoing percutaneous coronary intervention (PCI). The aim of present study is to access the prognostic significance of optimal platelet inhibition according to a given clinical state and ethnicity.
Methods: A total of 278 non high risk acute coronary syndrome (ACS) patients indicated for PCI were enrolled.Adenosine diphophate induced maximal platelet aggregation (ADP-PG max) was assessed with Lumi-Aggregometer by light transmission aggregometry method. The primary endpoint was the incidence of Thrombolysis in Myocardial Infarction (TIMI) defined bleeding outcome and significant entry-site complication within hospital and 6 months follow-up period. Blood was obtained for platelet aggregation 24h after PCI and 1 month follow up respectively. Receiver-operating characteristic (ROC) curve analysis was conducted to reveal the optimal platelet aggregation value defining enhanced clopidogrel responder for association of measurements with endpoints.
Results: A total of 24 patients (8.6%) met with primary endpoint in the study, while 4 (1.4%) TIMI major bleeding events, 11 (4.0%) minor bleeding events and 9 (3.2%) significant entry-site complications were observed. Follow-up ADP-PG max (OR=0.96, 95%CI, 0.93-0.99; p=0.008) and renal insufficiency (OR=3.38 95%CI, 1.26-9.19; p=0.02) were associate with the prediction of bleeding events. The optimal cutoff for follow up ADP-PG max was 24.5% [area under the curve 0.72 (95% confidence interval 0.59-0.85), p<0.001]. Bleeding occurred in 26.2% of patients with clopidogrel enhanced response (16/61), as compared with 3.7% of remaining patients (8/217), (hazard radio, 9.26; p<0.001).
Conclusions: In conclusion, enhanced clopidogrel responsiveness was associated with a higher risk of bleeding and entry-site complication. Platelet function test detected at an appropriate sampling time after clopidogrel administration may help identify high bleeding risk patients after coronary intervention.

GW25-e3234

Discrepancy in Measuring On-Clopidogrel Platelet Responsiveness by Vasodilator-Stimulated Phosphoprotein Phosphorylation and Platelet Aggregation Is Associated with Smoking Status Following ST-Elevation Myocardial Infarction

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Objectives: To investigate the potential mechanism accounting for the discrepancy of VASP phosphorylation and platelet aggregation (PAG) in evaluating high on-clopidogrel platelet reactivity in patients following ST segment-elevation myocardial infarction (STEMI).
Methods: 90 consecutive STEMI patients scheduled for emergency percutaneous coronary intervention (PCI) were enrolled. Platelet reactivity after clopidogrel loading dose (300 mg) was determined by two methods [platelet reactivity index (PRI), measured by vasodilator-stimulated phosphoprotein (VASP) phosphorylation flow